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Methylphosphonate (DIMP) at the Building E3640 Process Laboratory  
Site, Aberdeen Proving Ground-Edgewood Area, Aberdeen, Maryland

PRINCIPAL INVESTIGATOR: Dennis T. Burton, Ph.D.

CONTRACTING ORGANIZATION: University of Maryland at College Park  
Queenstown, Maryland 21658-0169

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The contractor, University of Maryland at College Park, hereby certifies that, to the best of its knowledge and belief, the technical data delivered herewith under Contract DAMD17-92-C-2066 is complete, accurate, and complies with all requirements of the contractor.

Date: October 31, 1999

Name and Title of Certifying Official:

  
Dennis T. Burton, Ph.D.  
Senior Research Scientist



## EXECUTIVE SUMMARY

Diisopropyl methylphosphonate (DIMP) was found in the vicinity of the Building E3640 Process Laboratory (Building E3640) site during the 1994-1995 Remedial Investigation/ Feasibility Study (RI/FS) for the Canal Creek Study Area of the Aberdeen Proving Ground - Edgewood Area, Aberdeen Proving Ground, Maryland. DIMP occurred in 13 of 15 subsurface soil samples (vertical extent of contamination was evaluated from below the top soil horizon to the water table) in the northeast corner of the site. The concentrations ranged from a minimum of 0.07 up to a maximum of 4.8 mg/kg (dry wt.).

DIMP was also found in two surficial wells downgradient of a surficial groundwater divide where the groundwater flow is to the north toward Kings Creek. The concentrations of DIMP were 0.08 and 2.17 mg/L in the surficial wells CCJ152A and CCJ153A, respectively. Subsequent analyses of three groundwater samples taken from well CCJ153A in 1998, showed that DIMP concentrations ranged from 4.72 to 6.05 mg/L.

The RI/FS concluded that 1) the DIMP in the subsurface soil is moving into the underlying surficial aquifer and 2) an apparent plume of DIMP is migrating in the surficial aquifer from the Building E3640 area towards Kings Creek. As a result, the U.S. Environmental Protection Agency (EPA) Region III recommended that additional information concerning the ecological hazard of DIMP be obtained for the site. A screening-level risk assessment was conducted to predict the likelihood of adverse ecological effects of DIMP in the subsurface soil at the site as well as the potential for adverse ecological effects as the compound moves in the surficial groundwater toward the Kings Creek area.

Two exposure pathways exist by which DIMP may reach the ecological receptors in the Building E3640 and Kings Creek areas. The first is the contaminated subsurface soil located in the spill area in the northeast corner of the Building E3640 site. The second potential exposure pathway is the apparent plume of DIMP which is migrating in the surficial aquifer from the Building E3640 area north northeast towards Kings Creek. All activities at Building E3640 which contaminated the soils and surficial aquifer were stopped in 1978. No DIMP is currently stored on site. Thus, no further releases of DIMP at the site will occur. Additional releases of DIMP to the groundwater could occur via the subsurface soils (infiltrating precipitation) in the spill area.

The following endpoints were used to evaluate the ecological risk of DIMP to the populations/communities in the Building E3640 and Kings Creek areas: 1) adverse effects to microorganisms, invertebrates, and plant communities from direct contact with DIMP in the soil; 2) adverse effects to aquatic life from exposure to DIMP in the sediment and water column; and 3) adverse effects to wildlife from the ingestion of material containing DIMP.

All no-observed-adverse-effect level (NOAEL) data used in the screening-level ecological risk assessment were based on conservative or worst case assumptions. A

major assumption was made that the surficial aquifer would ultimately transport DIMP to the palustrine wetlands and tidal wetlands of the Kings Creek area as well as Kings Creek. No evidence exists which shows that DIMP has in fact moved from the Building E3640 site. Likewise, the assumption was made that DIMP concentrations in the surface and subsurface soils in the palustrine wetland areas and tidal wetland sediments were the same as the highest concentration in the surficial aquifer since the surficial aquifer would be the source of the contaminant. To be conservative, 6.02 mg/kg was used as a worse case for all soil calculations even though the highest DIMP concentration in the subsurface soils at the Building E3640 site was 4.8 mg/kg. The bioconcentration data for plants (agricultural plants) reported in the literature and used in the risk assessment are questionable (data not treated statistically). However, the highest BCF (10.7) for agricultural plants was used for all plants in the risk assessment. The worst case assumption was made that DIMP was bioconcentrated to 64 mg/kg dry weight ( $10.7 \text{ [BCF]} \times 6.02 \text{ mg/kg} = 64$ ) in the roots, stems, and leaves of all plants in the Building E3640 and King Creek areas.

The assumption was made that the highest concentration of DIMP found in the surficial aquifer (6.02 mg/L) at the Building E3640 site was in equilibrium with the bulk sediment, sediment interstitial water and water column in Kings Creek. Thus, any organism present in Kings Creek was assumed to be exposed to a maximum concentration of 6.02 mg/L. The worse case assumption was made that the oral exposures for all wildlife in both the Building E3640 and Kings Creek areas would be 64 mg/kg/d. All wildlife found in the study area consume some plant material in their diet. Species which consume multiple food types, such as, a mixed diet of plants and animals would not consume DIMP at a rate of 64 mg/kg/d because animals do not accumulate DIMP above background levels.

A number of exposure-modifying factors (e.g., home range, season, behavior, etc.) can modify wildlife contaminant exposure. The assumption was made that all organisms were always in contact with the contaminant at the maximum concentrations given above. The assumption was also made that DIMP was 100% bioavailable to all receptors at all times. DIMP does not accumulate above background in animals because of its low log octanol water partition coefficient (1.03); thus, DIMP bioconcentration/ bioaccumulation in animals was not considered in the risk assessment. When chronic NOAEL data were not available for use in the risk assessment calculations, established uncertainty factors were used to estimate NOAELs from subchronic values.

No data are available regarding the modes of DIMP toxicity to organisms in most plant and animal phyla. Toxicokinetic studies have shown that DIMP is rapidly absorbed (15 min to 3 h depending on the species) following oral administration in mammals. DIMP is initially distributed throughout the body via the circulatory system, followed by high concentrations primarily in the liver, kidneys, and urinary bladder in 4 to 24 h after oral administration. DIMP is metabolized primarily to IMPA; some hydrolysis of IMPA to MPA may occur in the liver or in other tissues. Peak urinary excretion of a single dose occurs between 6 and 72 h depending on the species. No storage of DIMP, IMPA, or MPA occurs

in the body of mammals, although portions of [<sup>3</sup>H]-label may become incorporated in biomolecules leading to some retention of label in the form of unextractable labeled compound. DIMP is not genotoxic or carcinogenic to birds or mammals (including humans) after oral exposure. DIMP is not a developmental hazard to larval frogs.

Screening-level ecological risk calculations show that DIMP poses a negligible risk to plants and animals in the Building E3640 Process Laboratory site and Kings Creek areas. The conclusion was based on the fact that the hazard quotients (HQ) of all potential receptors in the study area were <1. The HQs for soil microorganisms, soil and litter invertebrates, and terrestrial plants were estimated to be 0.06, 0.05, and 0.60, respectively. The HQs for aquatic microorganisms (bacteria), aquatic algae, aquatic invertebrates, fish, and amphibians were 0.06, 0.01, 0.10, 0.04, and 0.02, respectively. The HQs for birds and mammals were 0.63 and 0.76, respectively. No DIMP data were available for reptiles; thus, an HQ was not calculated for reptiles. Based on the data for other vertebrates, the weight-of-evidence suggests that DIMP will not be a risk to reptiles.

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## LIST OF ACRONYMS AND ABBREVIATIONS

ACQUIRE	Aquatic Information Retrieval (ACQUIRE) Data Base
APG	Aberdeen Proving Ground
ASTM	American Society for Testing and Materials
BCF	Bioconcentration factor
CAS No.	Chemical Abstracts Service Registry Number
COR	Commanding Officer's Representative
DIMP	Diisopropyl methylphosphonate
DNAPL	Denser-than-water nonaqueous phase liquid
EC50	Concentration of a material that causes an effect other than death to 50% of the test organisms
EEC	Estimated environmental concentration
EPA	U.S. Environmental Protection Agency
GB	Sarin (isopropyl methylphosphonoflouridate)
HQ	Hazard quotient
IMPA	Isopropyl methylphosphonic acid
$K_{oc}$	Organic carbon partition coefficient
$K_{ow}$	Octanol water partition coefficient
LC50	Concentration of a material lethal to 50% of the test organisms
LD50	Dose of a material that is lethal to 50% of the test organisms
LOAEL	Lowest-observed-adverse-effect level
MPA	Methylphosphonic acid
MSL	Mean sea level
NOAEL	No-observed-adverse-effect level
PCBs	Polychlorinated biphenyl compounds
ppm	Parts per million (mg/L or mg/kg)
RI/FS	Remedial Investigation/Feasibility Study
SEM/AVS	Simultaneously extractable metal/acid volatile sulfide
TBP	Toxic burning pits
TI	Teratogenic index
UMD	University of Maryland
USACEHR	U.S. Army Center for Environmental Health Research
USGS	U.S. Geological Survey
VOCs	Volatile organic compounds
WBCC	West Branch of Canal Creek

## 1. INTRODUCTION

Diisopropyl methylphosphonate (CAS No. 1445-75-6) was found in the vicinity of the Building E3640 Process Laboratory (Building E3640) during the 1994-1995 Remedial Investigation/ Feasibility Study (RI/FS) for the Canal Creek Study Area of the Aberdeen Proving Ground-Edgewood Area (Jacobs Engineering Group Inc., 1995). DIMP occurred in 13 of 15 subsurface soil samples (vertical extent of contamination was evaluated to the water table) in the northeast corner of the site. The concentrations ranged from a minimum of 0.07 up to a maximum of 4.8 mg/kg (dry wt.). DIMP was also found in two surficial wells downgradient of a surficial groundwater divide where the groundwater flow is to the north toward Kings Creek. The concentrations of DIMP were 0.08 and 2.17 mg/L in the surficial wells CCJ152A and CCJ153A, respectively (Jacobs Engineering Group Inc., 1995). Subsequent analyses of three groundwater samples taken from well CCJ153A in March 1998, showed that DIMP concentrations ranged from 4.72 to 6.05 mg/L (Appendix 1).

The RI/FS concluded that 1) the DIMP in the subsurface soil is moving into the underlying surficial aquifer and 2) an apparent plume of DIMP is migrating in the surficial aquifer from the Building E3640 area towards Kings Creek (Jacobs Engineering Group Inc., 1995). As a result, EPA recommended that additional information concerning the hazard of DIMP be obtained for the site. A screening-level risk assessment was conducted to predict the likelihood of adverse ecological effects of DIMP in the subsurface soil at the site as well as the potential for adverse ecological effects as the compound moves in the surficial groundwater toward the Kings Creek area. The screening-level aquatic ecological risk assessment followed the EPA guidance procedures for Superfund sites (U.S. EPA, 1997). Additional guidance for ecological risk assessment was taken from EPA's Risk Assessment Forum final guidelines (U.S. EPA, 1998a).

## 2. SCREENING-LEVEL PROBLEM FORMULATION

### 2.1 Environmental Setting and Contaminants at the Site

The environmental setting and contaminants found in the vicinity of the Building E3640 Process Laboratory have been discussed in detail in the Remedial Investigation/Feasibility Study (RI/FS) report for the Canal Creek Study Area of the Aberdeen Proving Ground-Edgewood Area (Jacobs Engineering Group Inc., 1995). Briefly, the currently abandoned Building E3640, which is located on the north side of Beach Point Road, was constructed in 1951 and 1952 and operated through 1978 (Fig. 1). Most of the work at the site involved the preparation of materials or evaluation of production processes. Research involving the disposal of chemical agents was also performed at Building E3640. DIMP was used as a precursor in the scale-up of bench syntheses of the chemical warfare agent GB (Sarin)(Battelle, 1997).

Most liquid wastes generated at Building E3640 were discharged to the chemical sewer system (Fig. 1). Wastewater was collected from working bays in three sumps located below the floor on the north side of the building. Effluents from the sumps passed through a flow-through sump located northeast of the building and were discharged into an open ditch approximately 58 m (190 feet) northeast of Building E3640. Wastewater was then carried northward in the open ditch to a branch of Kings Creek. Chemicals and solid wastes were stored in drum racks northeast and west of Building E3640 as well as inside the building (Jacobs Engineering Group Inc., 1995).

The potential constituents of concern for the Building E3640 site include essentially all of the standard U.S. military chemical agents and post-World War II experimental agents. PCBs were used as heat transfer materials in the processes that were conducted on site. Other miscellaneous chemicals, such as B-1 dye, manufacturing raw materials, and intermediates of those materials, were used or stored on site (Jacobs Engineering Group Inc., 1995).

No chemical agents, including DIMP, were found in the surface soils at various spillage areas at the Building E3640 site. A number of other contaminants (e.g., metals, PCBs, pesticides, and herbicides) were found at various concentrations in the surface soils. DIMP was found in 13 of 15 subsurface soil samples in the spill area (~200 m<sup>2</sup>) located in the northeast corner of the site (Fig. 1). The vertical extent of contamination was evaluated from the bottom of the topsoil layer (topsoil horizon ranged from ~15 to 30 cm) to the water table. Thirteen of the subsurface samples were completed to a depth of 1.6 m (5 ft) while two were completed to a depth of 3.3 m (10 ft) since saturated conditions were encountered. The concentrations ranged from a minimum of 0.07 up to a maximum of 4.8 mg/kg (dry wt.). This suggests that wastewater discharged to the sewer line

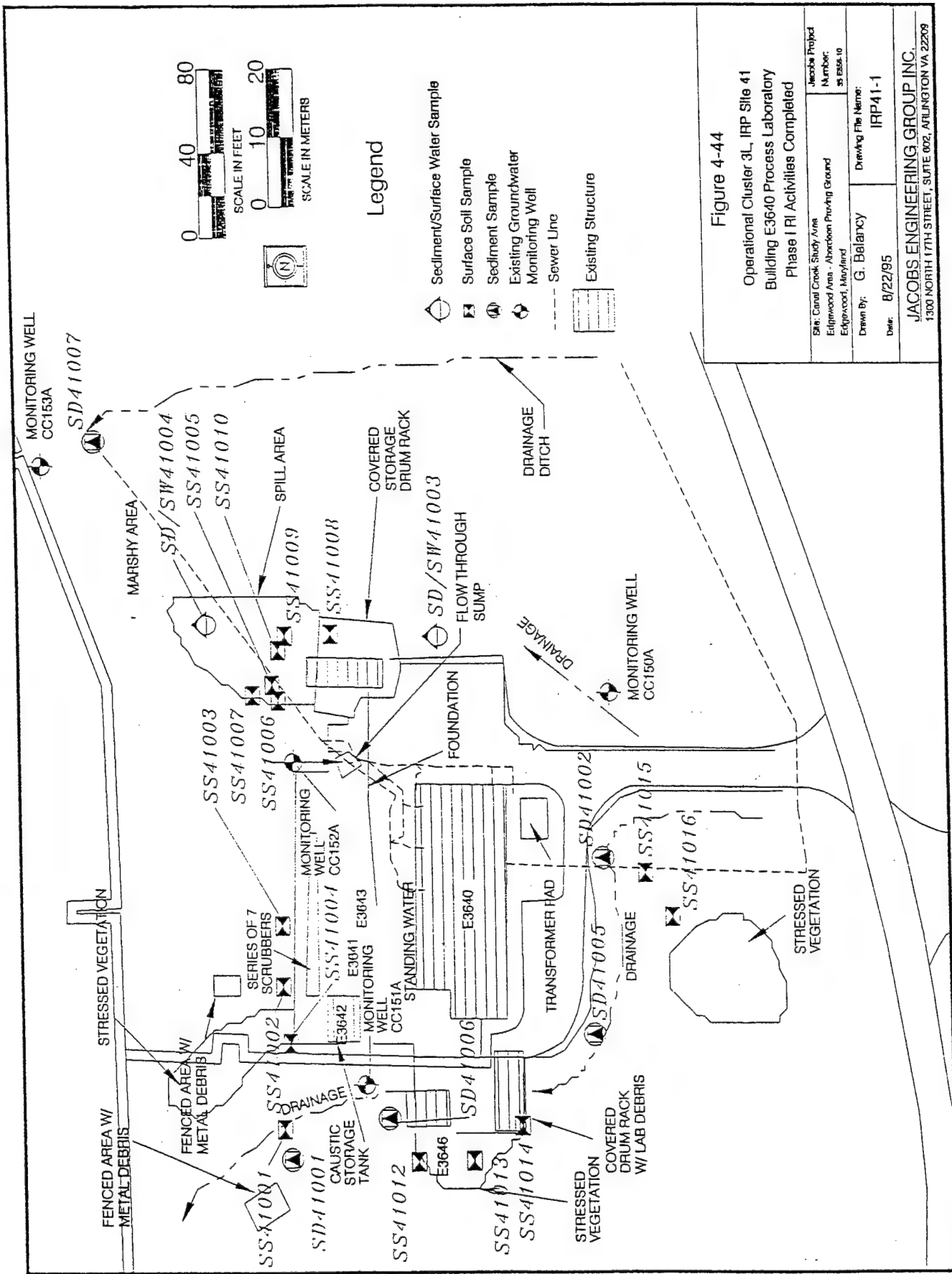


Figure 1. Schematic of the Building E3640 Process Laboratory site (Fig. taken from Jacobs Engineering Group Inc., 1995).

contained DIMP (Jacobs Engineering Group Inc., 1995). DIMP was not found in any sediments from the drainage ditches around the site, including the open ditch to Kings Creek. DIMP (0.0176 mg/L) was found in one surface water sample (SW41004) in the spill area where wastewater moved through the chemical sewer system to the open ditch going to Kings Creek. The site contains water only during wet periods of the year. According to Jacobs Engineering Group Inc. (1995), the presence of DIMP in the sample may be indicative that the surficial aquifer is in contact with the surface during wet periods of the year. No DIMP was found in a sediment sample (SD41004) taken at the same location.

DIMP has been found in two surficial wells downgradient of a surficial groundwater divide where the groundwater flow is to the north towards Kings Creek. DIMP concentrations of 0.077 and 2.170 mg/L were found in the surficial wells CCJ152A and CCJ153A, respectively (Jacobs Engineering Group Inc., 1995). Chemical analyses of the surficial groundwater taken from well CCJ153A on March 16, 18, and 20, 1998, documented concentrations of 6.05, 5.09, and 4.72 mg/L, respectively (Appendix 1). The RI/FS concluded that an apparent plume of DIMP is migrating in the surficial aquifer from the Building E3640 site towards Kings Creek (Jacobs Engineering Group Inc., 1995). The flow direction is based on the fact that Beach Point Road, which is located to the south of the site, is topographically high in the area and approximates a surficial groundwater divide. Thus, the surficial groundwater flow at the site should move to the north northeast towards Kings Creek. To the knowledge of the author of this report, no study has shown that the DIMP plume has in fact migrated from the source area into the Kings Creek area.

## **2.2 Contaminant Fate and Transport**

### **2.2.1 Hydrolysis**

The hydrolysis of DIMP in aqueous solution is very slow. The half-life of DIMP hydrolysis in groundwater obtained from the Intercept and Treatment System north of Rocky Mountain Arsenal (Commerce City, Colorado) has been estimated to be approximately 500 years when determined at elevated temperatures and extrapolated to 10°C (pH not given) (Sega et al., 1998). Bel'skii et al. (1969; as cited by Spanggord et al., 1979) estimated the half-life of DIMP in water at 10°C to be approximately 530 years (Bel'skii et al., 1969; as cited by Spanggord et al., 1979). The rate constants for DIMP hydrolysis in groundwater and ASTM Type II water are similar (Sega et al., 1998). According to Sega et al. (1998), this suggests that traces of metals and particulates in groundwater do not accelerate the rate of hydrolysis. The rates of DIMP hydrolysis in neutral aqueous solution have been shown to be much slower than those observed in either acidic or alkaline solution (Bel'skii et al., 1969; as cited by Sega et al., 1998).

The major hydrolysis products of DIMP in groundwater and surface water are isopropyl methylphosphonic acid (IMPA) (CAS No. 1832-54-8) and methylphosphonic acid (MPA) (CAS No. 993-13-5) (Sega et al., 1998; Spanggord et al., 1979). The hydrolysis products in groundwater were determined at 90°C over a 43-d period by Sega et al. (1998). According to Sega et al. (1998), the elevated study temperatures were mandatory because the very slow hydrolysis rate at ambient temperatures would have required extremely long periods of time to produce measurable quantities of hydrolysis products. The data of Sega et al. (1998) show that as DIMP was hydrolyzed under the conditions given above, IMPA concentration increased over time, but the MPA concentration was variable and did not increase steadily. The total concentration of DIMP and its products did not account for all of the DIMP that disappeared. At the end of the 43-d experiment, the molar accounting of unreacted DIMP and its detected hydrolysis products was 15% of the starting material. This consisted of 4% nonhydrolyzed DIMP, 9% IMPA, and 2% MPA. Inorganic phosphate was not detected. The authors stated that they could not offer an explanation for either the apparent lack of inorganic phosphate or the clear disparity in the molar accounting for DIMP and its hydrolysis products.

The hydrolysis rate of IMPA was also examined by Sega et al. (1998) using ASTM Type II water to avoid any interferences with phosphate in natural groundwater. The experiment was run at 90°C and observed for 71 d. IMPA hydrolyzed very slowly to MPA but at a rate 120 times slower than the hydrolysis rate of DIMP to IMPA at 90°C in ASTM Type II water. An extremely slow hydrolysis of MPA to inorganic phosphate has been reported by Schowanek and Verstraete (1991). Kingery and Allen (1995), in their review of the environmental fate of organophosphorus nerve agents, state that no hydrolysis products other than those discussed above have been reported in the literature.

### **2.2.2 Photolysis**

The photochemical transformation of DIMP was studied by Spanggord et al. (1979). The absorption spectrum from 280 to 700 nm showed that DIMP absorbs light weakly in the solar spectral region. DIMP (concentration not given) was not photolyzed in 9.7 d at >290 nm (UV-visible spectra) in either North Bog water taken from the Rocky Mountain Arsenal or distilled water. Thus, direct or indirect photolysis is not an important mechanism governing the environmental fate of DIMP in surface waters.

### **2.2.3 Volatilization**

DIMP adsorbs to soil; however, no experimentally determined soil partition coefficients could be found in the literature. Volatilization from soil is a slow process. Spanggord et al. (1979) showed that approximately 19% of the DIMP present in a DIMP-contaminated soil (2.7 mg/kg) from Rocky Mountain Arsenal volatilized as the parent compound over a 34 week period at 25°C. Van Voris et al. (1987) studied the persistence of DIMP deposited on the surface of two types of soil via aerosol

application. The DIMP-contaminated soils were maintained at 24°C for 14 d. The calculated half-lives for DIMP ranged from 26 to 28 d for the two soils. Van Voris et al. (1987) also studied the persistence of DIMP deposited on the surface of two types of plant leaves via aerosol application. The calculated half-lives for DIMP ranged between 3.6 and 4.2 d for the two foliar surfaces. Although not stated by the authors, it appears that the most likely process responsible for the loss of DIMP from the surface of soil and leaves was volatilization.

No experimental data could be found on the volatilization of DIMP from water. Estimates of the half-life of DIMP in water using Henry's Law constant ( $3.88 \times 10^6$  atm  $\text{m}^3/\text{mole}$ ; Meylan and Howard, 1991) were given in the Hazardous Substances Data Bank (HSDB, 1998). Using the calculations of Thomas (1990), the estimated volatilization half-life from a model river that is 1 m deep, flowing 1 m/sec with a 3 m/sec wind would be 12.8 d; the estimated half-life from a model lake (physical parameters not given) would be 97 d (HSDB, 1998).

According to data presented in the Hazardous Substances Data Bank, any DIMP present in the atmosphere would exist primarily in the vapor phase (HSDB, 1998). DIMP will react with photochemically produced hydroxyl radicals resulting in an estimated half-life of 5.2 h (HSDB, 1998).

#### **2.2.4 Bioconcentration**

Bioconcentration of DIMP by aquatic organisms and/or bioaccumulation by terrestrial organisms would not be expected to occur when one considers that the  $\log K_{ow}$  for DIMP is 1.03 (Krikorian et al., 1987). Bioconcentration of a material up to 100-fold above background (bioconcentration factor or BCF = 100) normally does not occur until  $\log K_{ow} = 3$  (U.S. EPA, 1991). Bentley et al. (1976) showed that DIMP did not bioconcentrate in bluegills exposed to 167 mg/L  $^{14}\text{C}$ -DIMP for 14 d. Toxicokinetic studies have shown that no storage of DIMP, IMPA, or MPA occurs in the tissues of mammals (ATSDR, 1998). As is the case for DIMP, no bioconcentration of IMPA or MPA would be expected to occur because the  $\log K_{ow}$ s for IMPA and MPA are 0.27 and -0.70, respectively (Meylan and Howard, 1995). Since DIMP and its major degradation products are not anticipated to bioconcentrate, no food chain bioaccumulation or transfer pathways are expected to be important in the fate and transport of the compounds in the environment.

#### **2.2.5 Biodegradation**

The biodegradation of DIMP by microorganisms in soil has been studied by Spanggord et al. (1979). The investigators showed that approximately 13.4% of the DIMP present in a DIMP-contaminated soil (2.7 mg/kg) from Rocky Mountain Arsenal was degraded by resident microorganisms to  $\text{CO}_2$  over a 34-week period at 25°C. The authors estimated that it would take more than 2 years for 50% of the parent compound to degrade to  $\text{CO}_2$  at 25°C. In a 17-week experiment at 10°C, <0.1% of the parent



compound degraded to  $\text{CO}_2$ . Thus, temperature is important in estimating the rate of biodegradation of DIMP in soil.

The biodegradation of DIMP by microorganisms in surface water has been studied by Spanggord et al. (1979). Organisms present in water taken from the North Bog at Rocky Mountain Arsenal, which contained  $\sim 0.26$  mg/L DIMP, were incubated with various nutrients added to the water at 10 and 25°C for 12 weeks. No detectable degradation of DIMP occurred in 12 weeks at either temperature. In a short-term study, Van Voris et. al. (1987) also showed that DIMP concentrations of  $\sim 25$  mg/L did not change over a 5-d period in freshwater laboratory microcosm tanks (pH = 5; alkalinity = 188 mg/L as  $\text{CaCO}_3$ ; temperature = 20°C).

The biodegradation of IMPA and MPA by microorganisms incubated with various nutrients in Rocky Mountain Arsenal soil and North Bog water was also studied by Spanggord et al. (1979). The authors showed that the microbes could readily split the carbon-phosphorus linkage of both compounds and thus use the molecules as a phosphate source. Spanggord et al. (1979) also grew microorganisms using IMPA and MPA as the sole phosphorus source in phosphate-deficient organic supplement media for soil and North Bog water. The investigators concluded that IMPA- and MPA-using microbes are present in the natural environment, and that the hydrolysis of DIMP to IMPA is the rate-limiting step in determining the environmental persistence of DIMP.

## **2.2.6 Transport in Soil and Groundwater**

A soil partition coefficient has not been experimentally determined for DIMP. The Hazardous Substances Data Bank provides data which estimate the  $K_{oc}$  for DIMP to be 111 and 31, respectively, when calculated via procedures using DIMP's  $\log K_{ow}$  and molecular structure (HSDB, 1998). According to the Hazardous Substances Data Bank, DIMP is highly mobility in soil. The aqueous solubility of DIMP ( $> 1$ g/L at 25°C) also suggests that DIMP should be mobile in soil and sediment (HSDB, 1998).

DIMP has been shown to be transported in groundwater. DIMP was discharged in industrial effluent at the Rocky Mountain Arsenal during the period 1952-1957 to unlined surface ponds (Robson, 1977). The compound moved through the underlying soils into a shallow alluvial aquifer and by 1974, it was found in a 73 km<sup>2</sup> area in the groundwater to the northwest of the arsenal (Robson, 1977).

## **2.3. Ecotoxicity and Potential Receptors**

### **2.3.1 Ecotoxicity**

#### **2.3.1.1 Toxicity to Microorganisms**

A literature search of several data bases revealed that no systematic studies



have been performed on the toxicity of DIMP to microorganisms. Biodegradation studies by Spanggord et al. (1979) provide indirect evidence that DIMP is not toxic to aquatic and soil microorganisms at concentrations up to 100 ppm. Microorganisms taken from North Bog water and soil obtained from the Rocky Mountain Arsenal were grown for biodegradation studies in basal salts medium with glucose and yeast extract at various DIMP concentrations up to 100 ppm. Comparisons of broth turbidity showed no growth inhibition up to 100 ppm DIMP (Spanggord et al., 1979).

#### **2.3.1.2 Toxicity to Soil and Litter Invertebrates**

The toxicity of DIMP to soil invertebrates was studied by Van Voris et al. (1987) using an earthworm (*Eisenia fetida*) as a representative invertebrate. A dose of 565 mg/kg dry soil did not affect 13 of 15 earthworms in a 14-d exposure. Two of the 15 earthworms were said to be "sluggish" after 14 d. A concentration of 4,011 mg/kg killed all earthworms by 14 days. Although no deaths occurred at the lower concentration, the authors estimated the 14-d LD50 to be ~1,500 mg/kg.

#### **2.3.1.3 Toxicity to Terrestrial Plants**

No quantitative data exist on the toxicity of DIMP applied to terrestrial plants in the root and soil zones which are the most likely routes of exposure to plants at the study site. Some data do exist for leaf surface exposures of DIMP via aerosols and sprays (Van Voris et al., 1987); however, the data will not be reported here since DIMP is not expected to contact the foliar surfaces of terrestrial plants via atmospheric deposition. O'Donovan and Woodward (1977a,b) conducted a qualitative study (no statistical analyses conducted) of the phytotoxicity of DIMP under both hydroponic and soil growth conditions. Eight agricultural and two horticultural plants were exposed to DIMP under hydroponic conditions. Five agricultural plants were exposed to DIMP under soil culture conditions. Both sets of experiments were conducted for periods up to five months. According to the investigators, phytotoxic symptoms in the hydroponic tests indicated that a phytotoxic effect occurred between 10 and 100 mg/L (no additional concentrations were studied between 10 and 100 mg/L). Severe tissue damage occurred in most plants above 100 mg/L. In the soil growth experiments, the investigators concluded that no phytotoxic effects occurred at concentrations up to 20 mg/L. The authors reported the concentration of DIMP in the irrigation water applied to the soil. No data were given showing the actual concentration retained in the soil. Rosenblatt et al. (1975) reported a range finding test which indicated that DIMP burned the leaf tips of two agricultural plants at aqueous concentrations of 10-40 mg/L (irrigation water applied to the soil). The potential mechanisms of toxicity were not discussed by O'Donovan and Woodward (1977a,b) or Rosenblatt et al. (1975).

O'Donovan and Woodward (1977a,b) also conducted a qualitative bioconcentration study with plants exposed to DIMP under the conditions given above. The investigators stated that DIMP was bioconcentrated to various degrees primarily in the leaves in most of the experimental plants. Less DIMP was bioconcentrated in the

stems and roots of the plants. No bioconcentration occurred in the leaves, stems, or roots of the juniper (species not given) over a 5-month period. The authors state in the abstract of the report that the BCFs were 20 or less in all the studies; however, no experimental data were presented to substantiate a BCF of 20. The highest BCF listed in the report for plants grown in soil was 10.7 for wheat leaves (species not given) exposed for 2.2 and 5 months to 8 mg/L DIMP in the irrigation water. The BCF for wheat leaves exposed for both 2.2 and 5 months at 20 mg/L DIMP was 5.3 mg/L. The BCF for wheat leaves grown at 1 mg/L for 2.2 months was <0.01. No data were available for wheat at 1 mg/L after 5 months of exposure. It is not clear why the BCFs at 8 mg/L were twice as high as those at 20 mg/L in both the 2.2 and 5 month exposures. The limited data given in O'Donovan and Woodward (1977a,b) indicate that the DIMP bioconcentrated in the tissues of various plants was not toxic to the plants.

#### 2.3.1.4 Toxicity to Aquatic Organisms

The acute toxicity of DIMP was established for several aquatic organisms by Bentley et al. (1976) and Van Voris et al. (1987). Burton and Turley (in review) conducted a study to determine the acute and chronic toxicity of DIMP and the possible interactions of DIMP with other contaminants that may be present in the surficial groundwater at the Building E3640 site as it enters the aquatic environment. The toxicity of DIMP and its interaction with other contaminants in well CCJ153A was investigated as a worst case condition in the surficial aquifer. The toxicity of the parent compound was also determined by Burton and Turley (in review) to confirm the acute toxicity data in the literature and to provide chronic toxicity data which were not available.

The acute toxicity of DIMP to aquatic organisms is summarized in Table 1. Acute toxicity data are available for five species of freshwater algae, five vertebrates, four fish, and one frog. The 96-h EC50s (reduction in cell density) for algae range from a low of 2,234 mg/L for the blue-green alga, *Microcystis aeruginosa*, up to 6,107 mg/L for the blue-green alga, *Anabeana flos-aquae*. The acute toxicity (48-h LC50s) for invertebrate ranges from a low of 267 mg/L for a daphnid up to 2,160 mg/L for the sowbug. The 96-h LC50s for fish range from a low of 285 mg/L for fingerling channel catfish up to 631 mg/L for young rainbow trout.

The 96-h LC50 for frog (*Xenopus laevis*) embryos exposed to DIMP is 1,543 mg/L (Table 1). The 96-h NOAEL and LOAEL for mortality are 398 and 569 mg/L, respectively. The 96-h EC50 for malformations is 1,225 mg/L. A NOAEL and LOAEL for malformations could not be determined because significant mortality occurred at exposure concentrations of 569 mg/L and above (Appendix 1). The teratogenic index (TI), which by definition is the 96-h LC50 divided by the 96-h EC50 (malformations), provides an estimate of the teratogenic risk associated with a material (Dumont et al., 1983). TI values of 1.5 to 2.0 indicate that a material may be a potential teratogen. Materials with TI values >2.0 should be considered for further teratogenicity testing. The TI in the Burton and Turley (in review) study was ~1.3; thus, a low potential exists

TABLE 1. SUMMARY OF THE DIMP TOXICITY DATA BASE FOR AQUATIC ALGAE, INVERTEBRATES, FISH, AND AMPHIBIANS<sup>a</sup>

Species	Endpoint	Toxicity Value (mg/L)	References (Footnotes)
<b>ACUTE TOXICITY</b>			
<b>Algae</b>			
Green ( <i>S. capricornutum</i> )	96-h EC50 <sup>b</sup>	3,185	c
		2,623	d
		>500 <sup>d</sup>	e
Green ( <i>C. pyrenoidosa</i> )	96-h EC50 <sup>b</sup>	>500 <sup>d</sup>	e
Blue-green ( <i>M. aeruginosa</i> )	96-h EC50 <sup>b</sup>	2,234	d
Blue-green ( <i>A. flos-aquae</i> )	96-h EC50 <sup>b</sup>	6,107	d
Diatom ( <i>N. pelliculosa</i> )	96-h EC50 <sup>b</sup>	2,345	d
<b>Invertebrates</b>			
Cladoceran	48-h LC50	610	c
Daphnid	48-h LC50	267	d
Midge	48-h LC50	1,720	d
Scud	48-h LC50	494	d
Sowbug	48-h LC50	2,160	d
<b>Fish</b>			
Fathead minnow	96-h LC50	604	c
		479	d
Bluegill	96-h LC50	406	d
Channel catfish	96-h LC50	285	d
Rainbow trout	96-h LC50	631	d
<b>Amphibian</b>			
African clawed frog	96-h LC50	1,543	c
	96-h EC50 <sup>e</sup>	1,225	c
	NOAEL <sup>f</sup>	398	c
	LOAEL <sup>f</sup>	569	c

TABLE 1. (CONTINUED)

Species	Endpoint	Toxicity Value (mg/L)	References (Footnotes)
<b>CHRONIC TOXICITY</b>			
<b>Alga</b>			
Green ( <i>S. capricornutum</i> )	NOAEL <sup>b</sup>	711	<sup>c</sup>
	LOAEL <sup>b</sup>	1,423	<sup>c</sup>
<b>Invertebrate</b>			
Cladoceran	7-d LC50	375	<sup>c</sup>
	NOAEL <sup>h</sup>	142	<sup>c</sup>
	LOAEL <sup>h</sup>	285	<sup>c</sup>
<b>Fish</b>			
Fathead minnow	7-d LC50	381	<sup>c</sup>
	NOAEL <sup>i</sup>	142	<sup>c</sup>
	LOAEL <sup>i</sup>	285	<sup>c</sup>
Bluegill	14-d Bioconcentration	>167 <sup>j</sup>	<sup>d</sup>

<sup>a</sup> Summaries of the common and scientific names of the plants and wildlife used in the report are given in Tables 2 and 3, respectively.

<sup>b</sup> Test endpoint- reduction in growth (cell density).

<sup>c</sup> Burton and Turley (in review).

<sup>d</sup> Bentley et al. (1976).

<sup>e</sup> 500 mg/L DIMP highest concentration studied (Van Voris et al., 1987).

<sup>f</sup> Test endpoint- increase in malformations.

<sup>g</sup> Test endpoint- mortality.

<sup>h</sup> Test endpoint- reduction in neonate production.

<sup>i</sup> Test endpoint- reduction in growth.

<sup>j</sup> 167 mg/L DIMP highest concentration studied; no apparent stress or bioconcentration of DIMP occurred during a 14-d exposure (Bentley et al., 1976).

**TABLE 2. COMMON AND SCIENTIFIC NAMES OF THE PLANT  
SPECIES USED IN THE TEXT**

<b>Major Groups</b>	<b>Common Name</b>	<b>Scientific Name</b>
<b>Algae</b>	Blue-green	<i>Microcystis aeruginosa</i>
	Blue-green	<i>Anabeana flos-aquae</i>
	Diatom	<i>Navicula pelliculosa</i>
	Green	<i>Selenastrum capricornutum</i>
	Green	<i>Chlorella pyrenoidosa</i>
<b>Emergent Beds</b>	Arrow arum	<i>Peltandra virginica</i>
	Pickerelweed	<i>Pontederia cordata</i>
<b>High Marsh</b>	Common winterberry	<i>Ilex verticillata</i>
	Dense-flower smartweed	<i>Polygonum densiflorum</i>
	Marsh mallow	<i>Althaea officinalis</i>
	Narrow-leaved cattail	<i>Typha angustifolia</i>
	Northern wild rice	<i>Zizania aquatica</i>
	Southern wild rice	<i>Zizaniopsis milacea</i>
	Walter's millet	<i>Echinochloa walteri</i>
<b>Herbs</b>	Arrowhead	<i>Sagittaria</i> spp.
	Barberry	<i>Berberis</i> spp.
	False nettle	<i>Boehmeria cylindrica</i>
	Ferns	<i>Thelypteris</i> spp.
	Harlberd-leaved tearthumb	<i>Polygonum arifolium</i>
	Jack-in-the-plpit	<i>Arisaema triphyllum</i>
<b>Shrubs</b>	Cranberry	<i>Vaccinium</i> spp.
	Sassafras	<i>Sassafras albidum</i>
<b>Understory</b>	American holly	<i>Ilex opaca</i>
	Dogwood	<i>Cornus florida</i>
	Tulip popular	<i>Liriodendron tulipifera</i>
<b>Canopy</b>	Black gum	<i>Nyssa sylvatica</i>
	Hickories	<i>Carya</i> spp.
	Oaks	<i>Quercus</i> spp.
	Red maple	<i>Acer rubrum</i>
	Sweet gum	<i>Liquidambar styraciflua</i>

**TABLE 3. COMMON AND SCIENTIFIC NAMES OF THE WILDLIFE  
SPECIES USED IN THE TEXT**

<b>Major Groups</b>	<b>Common Name</b>	<b>Scientific Name</b>
<b>Invertebrates</b>	Cladoceran	<i>Ceriodaphnia dubia</i>
	Daphnid	<i>Daphnia magna</i>
	Earthworm	<i>Eisenia fetida</i>
	Midge	<i>Chironomous tentans</i>
	Scud	<i>Gammarus fasciatus</i>
	Sowbug	<i>Asellus militaris</i>
<b>Fish</b>	Atlantic menhaden	<i>Brevoortia tyrannus</i>
	Black drum	<i>Pogonias cromis</i>
	Bluegill	<i>Lepomis macrochirus</i>
	Carp	<i>Cyprinus carpio</i>
	Channel catfish	<i>Ictalurus punctatus</i>
	Fathead minnow	<i>Pimephales promelas</i>
	Gizzard shad	<i>Dorsoma cepedanum</i>
	Killifish	<i>Fundulus</i> spp.
	Rainbow trout	<i>Oncorhynchus mykiss</i>
	White perch	<i>Morone americana</i>
	Yellow perch	<i>Perca flavescens</i>
<b>Amphibians</b>	African clawed frog	<i>Xenopus laevis</i>
	Frogs	<i>Rana</i> spp.
<b>Reptiles</b>	Eastern box turtle	<i>Terrepene carolina</i>
	Eastern painted turtle	<i>Chrysemys picta</i>
<b>Birds</b>	Mallard duck	<i>Anas platyrhynchos</i>
<b>Mammals</b>	Eastern chipmunk	<i>Tamias striatus</i>
	White-tailed deer	<i>Odocoileus virginianus</i>
	Eastern grey squirrel	<i>Sciurus carolinensis</i>
	Mink	<i>Mustela vison</i>
	Muskrat	<i>Ondatra zibethicus</i>
	New Zealand white rabbit	<i>Oryctolagus cuniculus</i> .
	Opossum	<i>Didelphis marsupialis</i>
	Rat (Sprague-Dawley)	<i>Rattus norvegicus</i>
	Red fox	<i>Vulpes vulpes</i>

that DIMP is a developmental hazard.

Short-term chronic data are available for a freshwater green alga (growth), cladoceran (survival and reproduction), and a larval fish (survival and growth)(Table 1). The LOAEL and NOAEL (cell density) for the green alga *Selenastrum capricornutum* are 1,423 and 711 mg/L, respectively. The LOAEL (reduction in neonate production) for the invertebrate *Ceriodaphnia dubia* is 285 mg/L; the NOAEL is 142 mg/L. The larval fathead minnow LOAEL and NOAEL (reduction in growth) are 285 and 142 mg/L, respectively. Bentley et al. (1976) conducted a bioconcentration study with bluegill exposed to 167 mg/L  $^{14}\text{C}$ -DIMP for 14 d followed by a 7-d depuration phase. According to the authors, the bluegill appeared normal, fed readily, and generally showed no signs of stress during the study. No bioconcentration of DIMP occurred in the study. As discussed in Section 2.2.4, bioconcentration of DIMP would not be expected when one considers that the  $\log K_{ow}$  is 1.03.

Burton and Turley (in review) conducted an aquatic study to determine the acute and chronic toxicity of DIMP and the possible interactions of DIMP with other contaminants that may be present in the surficial groundwater at Building E3640. The toxicity of DIMP and its interaction with other contaminants in well CCJ153A was investigated as a worst case condition in the surficial aquifer. The groundwater was not acutely toxic to a green alga, cladoceran or larval fathead minnow. The groundwater was not acutely toxic to frog embryos (*Xenopus laevis*) after 96 h of exposure. A statistically significant ( $\alpha = 0.05$ ) effect was found for frog embryo malformations; however, the effect was judged to be statistical error because the concentrations in the groundwater were more than two orders of magnitude lower than the LOAEL (malformation effect) established for the parent compound. The acute values for the alga, invertebrate, and larval fish species in the study fell within the range of acute values established in the study by Bentley et al (1976) for several other freshwater species (Table 1). As was the case for acute toxicity, the groundwater did not cause any short-term chronic toxicity to a green alga, cladoceran, or larval fish.

The concentrations of DIMP in the three surficial groundwater samples (well CCJ153A) used in the toxicity studies by Burton and Turley (in review) ranged from 4.72 to 6.05 mg/L. Low concentrations of several priority pollutant heavy metals (aluminum, barium, chromium, copper, and manganese) and one volatile organic (vinyl chloride) were also present in one or more of the groundwater samples. No base neutrals, acid extractables, organophosphorus pesticides, or chlorinated pesticides and herbicides were found in the groundwater above the detection limits for drinking water. No nitroaromatic or nitramine munitions above a detection limit of 50  $\mu\text{g/L}$  were present. No analyses were conducted for IMPA and MPA. Because the groundwater was not acutely or chronically toxic to species from four trophic levels, the interactions of the metals, vinyl chloride, IMPA and MPA (if present) with DIMP were eliminated for further consideration in the screening-level risk assessment.



A search of EPA's ACQUIRE data base showed that no data exist concerning the mode of toxic action for DIMP to aquatic organisms (U.S. EPA, 1999). As discussed above, a low potential exists that DIMP is a developmental hazard. The low  $K_{ow}$  for DIMP indicates that the compound will not be bioaccumulated in animals. Likewise, no toxicity data for aquatic organisms were found for IMPA or MPA using EPA's ACQUIRE data base (U.S. EPA, 1999). The lack of toxicity data for a known material in EPA's ACQUIRE data base frequently means that the material is not toxicologically important (Norberg-King, 1999). As discussed above, IMPA or MPA are not expected to bioaccumulate because of the compound's low log  $K_{ow}$ s.

### 2.3.1.5 Toxicity to Wildlife

Wildlife may be exposed to DIMP through oral ingestion, inhalation, and dermal absorption. The most probable route at the study site is oral ingestion of food (either plant or animal) and water. In addition, some animals may ingest soil incidentally while foraging to meet nutritional needs. The risk of exposure via inhalation is small since DIMP does not readily volatilize (Sect. 2.2.3). No DIMP inhalation studies have been reported in the literature for any animals including humans (ATSDR, 1998; EPA, 1998b). Dermal absorption of DIMP is possible; however, with the possible exception of animals that forage in water, dermal exposure to DIMP is small relative to exposure via oral ingestion. One report was found concerning dermal exposure in New Zealand white rabbits. Hart (1976) established a LD50 of 1,100 mg/kg for dermal toxicity to the New Zealand white rabbit when DIMP was applied once to several areas on the back of the animals.

No DIMP toxicity data were found in the literature for amphibians or reptiles with the exception of the frog study discussed above. Two acute and one subchronic oral toxicity studies were found in the literature for birds exposed to DIMP; no chronic exposure data were found for birds (Table 4). The oral LD50 for the mallard duck is 1,490 mg/kg/d for a single dose (Aulerich et al., 1979; as cited in ATSDR, 1998). Blood pressure decreased in mallards given a single dose of 1,500 mg/kg/d via proventricular intubation (Jones et al., 1992). Pulse pressure and heart rate were not affected. Jones et al. (1992) speculated that DIMP acts by depressing or blocking nerve impulse transmission at the interneuron level of the central nervous system in the duck. Aulerich et al. (1979; as cited in ATSDR, 1998) also exposed the mallard for eight days to DIMP via oral administration. The NOAEL (no change in body weight) for the 8-d subchronic exposure was 1,007 mg/kg/d.

A number of acute oral studies have been conducted with various mammals exposed to DIMP. The acute responses include death, systemic, and neurological changes. The data have been summarized by the Agency for Toxic Substances and Disease Registry (ATSDR, 1998) and thus will not be given in this report. Likewise, several subchronic oral studies have also been conducted with endpoints which include systemic, immunological/lymphoreticular, neurological, reproductive, and developmental responses in various animals. The subchronic data have been summarized in reports by



**TABLE 4. DIMP TOXICITY TO BIRDS AND MAMMALS<sup>a,b</sup>**

Species	Frequency	NOAEL (mg/kg/d)	LOAEL (mg/kg/d)	Endpoint
<b>ORAL - ACUTE AND SUBCHRONIC EXPOSURE</b>				
<b>Birds</b>				
Mallard	Once		1,490	LD50 <sup>c</sup>
	Once		1,500	3/12 Died <sup>d</sup>
	Once		1,500	Decrease in blood pressure <sup>d</sup>
	8 d	1,007		No change in body weight <sup>c</sup>
<b>ORAL - CHRONIC EXPOSURE</b>				
<b>Mammals</b>				
Mink	49 weeks	95		Reproduction <sup>c</sup>
	13 months	330		Reproduction- 2 generations <sup>e</sup>
Rat	30 weeks	300		Reproduction- 3 generations <sup>f</sup>
	6-15 d	300		Developmental (gestation) <sup>f</sup>

<sup>a</sup> A summary of the common and scientific names of the wildlife used in the report is given in Table 3 of this report.

<sup>b</sup> No chronic data exist for birds. A number of oral subchronic exposure data exist for mammals; however, the data are not included in the table (see text).

<sup>c</sup> Aulerich et al. (1979; as cited in ATSDR, 1998).

<sup>d</sup> Jones et al. (1992).

<sup>e</sup> Bucci et al. (1997).

<sup>f</sup> Hart (1980).

the Agency for Toxic Substances and Disease Registry (ATSDR, 1998) and EPA's Integrated Risk Information System (U.S. EPA, 1998b); thus, they will not be repeated in this report. Two chronic oral studies have been conducted with mammals which examined reproduction and development (Table 4). Emphasis will be placed on these studies, as recommended by Sample et al. (1996), because the endpoints may be directly related to potential population-level effects. Although reproduction and development may have direct

implications on the viability of populations, population-level effects are not being evaluated in this screening-level ecological risk assessment.

Hart (1980) conducted a three generation reproductive study of rats. The following summary of Hart's (1980) study was taken from the Agency for Toxic Substances and Disease Registry (ATSDR, 1998):

In a three generation reproductive study, male and female rats received diisopropyl methylphosphonate in their feed (0, 30, or 300 mg/kg/day) for 11 weeks prior to being mated with animals of the same dose group in the 12th week. Dosing continued during gestation and lactation. A week after lactation of the  $F_{1A}$  pups, the  $F_0$  females were remated with a different male. A week after lactation of the  $F_{1B}$  pups, the  $F_0$  parents were sacrificed and necropsied. Male and female  $F_{1B}$  animals were selected and mated as above. Similarly  $F_{2B}$  offspring were mated, yielding third generation ( $F_{3A}$  and  $F_{3B}$ ) offspring. No differences in male virility or female fertility were noted in the  $F_0$  and  $F_1$  parents, and no differences in newborn viability or pup weights were noted in the  $F_1$  and  $F_2$  offspring. A significant number of pup losses were noted in the  $F_{3A}$  offspring from the 300 mg/kg/day group; however, since the losses were not observed in the second mating ( $F_{3B}$  offspring), the losses were probably not treatment related. Further, pup appearance and gross examination at necropsy did not reveal any evidence of diisopropyl methylphosphonate related effects in the  $F_{3A}$  and  $F_{3B}$  pups, although the histopathological changes were apparently not evaluated. No significant differences in body weight and food consumption among the  $F_0$  (parent),  $F_{1B}$ , and  $F_{2B}$  generations were observed. Necropsy observations did not indicate any dose-dependent relationships of diisopropyl methylphosphonate in the feed at doses of 30 and 300 mg/kg/day in the rat in three successive generations with two matings per generation.

Reproductive toxicity in mink has been studied over a 49-week period by Aulerich et al. (1979; as cited in ATSDR, 1998) and a 13-month period study by Bucci et al. (1997). The following summary was taken from the Agency for Toxic Substances and Disease Registry (ATSDR, 1998):

Reproductive toxicity in mink was assayed in a 49-week feed study (Aulerich et al., 1979). Male and female dark variety mink received feed containing diisopropyl methylphosphonate at doses of 0, 11, 37, or 95 mg/kg/day. Male fertility, estimated by the presence of sperm in post-coital vaginal aspirations,

was not adversely affected. Further, no significant differences were noted in whelping dam and kit performance, kit mortality, kit weight, or the body weight of lactating females at 4 weeks post-partum (Aulerich et al., 1979). In the study, an increase in deaths occurred in females that was statistically significant at the high dose. No control females died, while 2 of 23, 3 of 24, and 5 of 224 died at the low, middle, and high doses, respectively. However, the deaths may not be treatment related. In a concurrent study conducted to assess the toxicity of dicyclopentadiene which used mink from the same lot, the mortality in the unrelated female mink was 4 of 24 with 2 mink dying between the time of mating and lactation.

The conclusion that female deaths in the Aulerich et al. (1979) study were probably not DIMP treatment-related was supported by a two-generation reproductive study performed using Ranch Wild mink fed 0, 16, 45, or 262 mg/kg/day (males) or 0, 20, 57, or 330 mg/kg/day (females) diisopropyl methylphosphonate in the diet (Bucci et al., 1997). The  $F_0$  males and females were exposed for 1 and 4 months, respectively, and the  $F_1$  males and females for 8 and 13 months, respectively. No treatment-related effects were observed in kits/litter, live kits/litter, weights at birth or at 28 days, or kit development. No dose-related death was observed in females in either the  $F_0$  and  $F_1$  generations. Ovarian follicles were counted in control and high-dose  $F_1$  females to examine possible ovarian toxicity. There was a significant ( $p < 0.01$ ) increase in the mean follicle count of high-dose females ( $645 \pm 157$ ) compared to controls ( $329 \pm 153$  or  $460 \pm 148$ ). Only the control and high-dose animals' ovaries were examined. However, it is not clear whether this end point represents an adverse effect because the treated dams of both the  $F_0$  and  $F_1$  generations produced as many offspring as controls. The study authors noted that the effect could be representative of disrupted follicle maturation with retention of ova. Semen quality in  $F_0$  and  $F_1$  males, as measured by sperm motility, epididymal sperm count, and incidence of head/tail abnormalities, was unaffected by treatment.

Toxicokinetic studies have shown that DIMP is rapidly absorbed (15 min to 3 h depending on the species) following oral administration in animals. DIMP is initially distributed throughout the body via the circulatory system, followed by high concentrations primarily in the liver, kidneys, and urinary bladder in 4 to 24 h after oral administration (ATSDR, 1998). DIMP is metabolized primarily to IMPA; some hydrolysis of IMPA to MPA may occur in the liver or in other tissues (ATSDR, 1998). Peak urinary excretion of a single

dose occurs between 6 and 72 h depending on the species (ATSDR, 1998). No storage of DIMP, IMPA, or MPA occurs in the body, although portions of [<sup>3</sup>H]-label may become incorporated in biomolecules leading to some retention of label in the form of unextractable labeled compound (ATSDR, 1998). No studies were reported in the Agency for Toxic Substances and Disease Registry, Integrated Risk Information System, or the Hazardous Substances Data Bank regarding genotoxic effects in mammals (including humans) after oral exposure to DIMP (ATSDR, 1998; EPA, 1998b; HSDB, 1998). No studies were reported in the Agency for Toxic Substances and Disease Registry, Integrated Risk Information System, or Hazardous Substances Data Bank regarding carcinogenic effects in mammals (including humans) after oral exposure to DIMP (ATSDR, 1998; EPA, 1998b; HSDB, 1998).

### 2.3.2 Potential Receptors

The potential receptors for DIMP are the mixed sweet gum and subsoil communities at the site of contamination in the Building E3640 area. It is conceivable that the vegetation in the palustrine and tidal wetlands which border the western portion of Kings Creek watershed could be exposed to DIMP if one assumes that the surficial aquifer moves into these areas. Wildlife that feed on terrestrial vegetation in the Building E3640, palustrine, and tidal wetlands areas could be exposed to DIMP that has bioconcentrated in the plants. Likewise, wildlife could be exposed to DIMP if the surficial aquifer moves through the surface soils in the palustrine and wetland areas. If this was to occur, it would most likely occur during the wet seasons of the year. Sediment and aquatic communities of Kings Creek could be exposed to DIMP if the groundwater plume ultimately moves into the creek. Likewise, terrestrial species foraging on food items in Kings Creek could be exposed to DIMP.

The composition of the mixed sweet gum forest, which was taken from Garcia et al. (1995), consists of the following. The canopy is dominated by sweet gum, oaks, black gum, tulip popular, hickories, and red maple. The understory consists of dogwood, American holly, and sassafras. Barberry and cranberry are the dominant shrubs found in the mixed sweet gum community. The dominant herbs are grasses, ferns, and jack-in-the-pulpit. Grosbeaks, finches, fruit-eating songbirds, upland game birds, chipmunk, red fox, grey squirrel, opossum, and white-tailed deer are also found in the mixed sweet gum forest (Garcia et al., 1995). No information exists on the soil communities in the Building E3640 area.

Cranberry is the dominant shrub in the palustrine wetlands (Garcia et al., 1995). The following herbs can be expected to occur in the palustrine wetlands: false nettle, heart-leaved tearthumb, ferns, and arrowhead. The wildlife consists of freshwater invertebrates and amphibians. No information exists on the soil community in the palustrine wetlands.

The tidal wetland communities along the banks of Kings Creek consist of a number of species (Garcia et al., 1995). The high marsh community consists of narrow-leaved

cattail, northern and southern wild rice, Walter's millet, common winterberry, marsh mallow, and dense-flower smartweed. The emergent beds consist of pickerweed and arrow arum. The following wildlife may be found: muskrat, several species of birds, turtles, frogs, and freshwater and brackish water invertebrates and fish.

Kings Creek provides aquatic habitat for a variety of freshwater and estuarine species. Fish species that have been caught in Kings Creek include carp, channel catfish, Atlantic menhaden, black drum, white perch, yellow perch, and gizzard shad (Ehlers et al., 1995). Killifish have been observed by the author of this report in the shallow areas of the Creek. Invertebrates that occur in the sediments include polychaetes, oligochaetes, isopods, amphipods, and a variety of freshwater insects. A variety of estuarine invertebrates including clams and isopods are also likely to occur in the sediment of Kings Creek (Ehlers et al., 1995). No submerged aquatic vegetation beds have been observed in the near-shore areas, though they may exist in the main body of the Creek (Garcia et al., 1995).

## **2.4 Complete Exposure Pathways**

Two exposure pathways exist by which DIMP may reach the ecological receptors discussed above. The first is the contaminated subsurface soil located in the spill area in the northeast corner of the Building E3640 site. The second potential exposure pathway is the apparent plume of DIMP which is migrating in the surficial aquifer from the Building E3640 area north northeast towards Kings Creek. All activities at Building E3640 which contaminated the soils and surficial aquifer were stopped in 1978. No DIMP is currently stored on site. Thus, no further releases of DIMP at the site will occur. Additional releases of DIMP to the groundwater could occur via the subsurface soils (infiltrating precipitation) in the spill area.

The potential ecological receptors for DIMP in the subsurface soils located at the Building E3640 site are the mixed sweet gum forest community and soil-dwelling species that may move into or live in the subsurface soils. The exposure route for the mixed sweet gum plant community would be via the roots of the plants located in the subsurface soil. Qualitative data indicate that DIMP may be bioconcentrated in the leaves of agricultural plants. Although no evidence exists that bioconcentration may occur in woody plants, the assumption is being made that DIMP could be absorbed from the subsurface soil via the roots and subsequently bioconcentrated in the plants. Thus, the conservative assumption is being made that wildlife that consumes terrestrial plants will be exposed to DIMP. Exposure to DIMP via volatilization to the atmosphere (inhalation) is low because DIMP is not found in the surface soils. The exposure route for the subsurface terrestrial species would be via ingestion and dermal absorption.

The potential receptors for DIMP in the surficial aquifer would be the organisms located in the palustrine wetlands, tidal wetlands, and Kings Creek if one assumes that the contaminated groundwater plume ultimately reaches those areas. The possible receptors in the palustrine wetland areas would be those plants that come into contact with the

surficial aquifer via their root system. Freshwater invertebrates, amphibians, and other wildlife could be exposed to DIMP if the surficial aquifer moved through the surface soils into the wetland during wet periods of the year. The potential receptors in the tidal wetland areas would be those plants and wildlife exposed during intermittent flooding associated with normal action of the tides. Exposure could also occur if the surficial aquifer moved through the sediments. Both the sediment and water column organisms in Kings Creek could be exposed as the groundwater moved into the system. Likewise, terrestrial wildlife could be exposed while foraging on food items from Kings Creek.

## **2.5 Assessment and Measurement Endpoints**

Assessment endpoints are explicit expressions of the actual environmental value that is to be protected, operationally defined by an ecological entity and its attributes (U.S. EPA, 1997). All ecosystems are diverse, with many levels of ecological organization (e.g., individuals, populations, communities, ecosystems, and landscapes). It is rarely clear which of these characteristics are most critical to ecosystem function (U.S. EPA, 1998a). Ecologically relevant endpoints may be identified at any level of organization. Individual assessment endpoints usually encompass a group of species or populations with some common characteristics, such as, a specific exposure route or contaminant sensitivity (U.S. EPA, 1997). The consequences of changes in these endpoints may be quantified (e.g., alteration of community structure from loss of a keystone species) or inferred (e.g., survival of individuals to maintain populations) (U.S. EPA, 1998a).

The following endpoints will be used to evaluate the ecological risk of DIMP to the populations/communities described in Section 2.4.

- Adverse effects to microorganisms, invertebrates, and plant communities from direct contact with DIMP in the soil;
- Adverse effects to aquatic life from exposure to DIMP in the sediment and water column; and
- Adverse effects to wildlife from the ingestion of material containing DIMP.



### 3. SCREENING-LEVEL EXPOSURE ESTIMATE AND RISK CALCULATION

#### 3.1 Screening-Level Exposure Estimates

The highest measured or estimated on-site contaminant concentration for each environmental medium is normally used to estimate exposures (U.S. EPA, 1997). The highest concentration of DIMP found in the subsurface soils in the spill area at Building E3640 is 4.8 mg/kg dry weight. The assumption is being made that DIMP concentrations in the surface and subsurface soils in the palustrine wetland areas and tidal wetland sediments will be the same as the highest concentration in the surficial aquifer since the surficial aquifer would be the source of the contaminant. To be conservative, 6.02 mg/kg will be used as a worst case for all soil calculations even though the concentration at the Building E3640 site is lower. The bioconcentration data for plants are not very reliable; however, the worst case assumption will be made that DIMP will bioconcentrate up to 64 mg/kg dry weight ( $10.7 \text{ [BCF]} \times 6.02 \text{ mg/kg} = 64$ ) in the roots, stems, and leaves of all plants in the Building E3640 and King Creek areas. The use of a BCF of 10.7 is a more conservative estimator of plant concentration than a BCF of 1 which is typically recommended by EPA Region III BTAG for a screening-level ecological risk assessment (Elias, 1999).

A soil/sediment partition coefficient for DIMP could not be found in the literature (Sect. 2.2.6). According to the Hazardous Substances Data Bank, DIMP is highly mobile in soil (HSDB, 1998). It is unlikely that significant amounts of DIMP would partition into King Creek sediments if the surficial groundwater plume ultimately moved into the creek. A worst case assumption is being made that the highest concentration found in the groundwater (6.02 mg/L) will be in equilibrium with the bulk sediment, sediment interstitial water and water column. The assumption is being made that DIMP is 100% bioavailable to all receptors. DIMP is not expected to bioaccumulate in animals because of its low log  $K_{ow}$  (1.03); thus, DIMP is not important from a dietary standpoint.

#### 3.2 Screening-Level Risk Calculations

The hazard quotient approach is recommended for the screening-level risk calculations for Superfund sites (U.S. EPA, 1997). The hazard quotient (HQ) can be expressed as the ratio of a potential exposure level to the NOAEL:

$$\text{HQ} = \text{EEC} \div \text{NOAEL} \quad \text{or} \quad \text{HQ} = \text{Dose} \div \text{NOAEL}$$

where

EEC = estimated environmental concentration at the site, and

Dose = estimated contaminant intake at the site.

An HQ <1 (unity) indicates that the contaminant alone is unlikely to cause adverse ecological effects. As stated in the Superfund ecological risk assessment guidance document (U.S. EPA, 1997), an HQ <1 does not indicate the absence of ecological risk; rather, it should be interpreted based on the severity of the effect and the calculated hazard quotient. As certainty in the exposure concentrations and the NOAEL increase, there is greater confidence in the predictive value of the quotient method, and unity becomes a more certain pass/fail decision point. The HQs for the three endpoints listed in Section 2.5 are as follows:

### **3.2.1 Adverse Effects to Microorganisms, Invertebrates, and Plant Communities from Direct Contact with DIMP in the Soil**

The biodegradation studies by Spanggard et al. (1979) provide indirect evidence that DIMP is not toxic to soil microorganisms at concentrations up to 100 mg/kg (Sect. 2.3.1.1). Thus, one may assume that the NOAEL would be at least 100 mg/kg for soil microorganisms. The hazard quotient for soil microorganisms exposed to DIMP would be 0.06 (6.02 mg/kg ÷ 100 mg/kg) (Table 5). An HQ of 0.06 indicates that DIMP is unlikely to cause an adverse ecological effect to microbial communities.

The only DIMP data available for soil invertebrates are for the earthworm. According to Edwards (1992), earthworms are very important soil invertebrates because of their activity in promoting soil fertility. Their feeding and burrowing activities break down organic matter and release nutrients and improve aeration, drainage, and aggregation of soil. Earthworms are exposed to contaminants via dermal and oral routes. Because of its importance, the earthworm is the most frequently used invertebrate for estimating risk to soil and litter invertebrates (Efroymson et al., 1997a).

Van Voris et al. (1987) calculated an acute 14-d LD50 of ~1,500 mg/kg dry soil for the earthworm; however, the estimate was based on two data points. Thus, confidence in the value is low because of the limited number of concentrations. The authors did show that concentrations of 565 mg/kg dry soil did not cause any mortality to the earthworm in acute exposures. Efroymson et al. (1997a) have shown that an uncertainty factor of 5 can be applied to acute LD50 data for earthworms exposed to organic compounds to estimate safe thresholds for growth and reproduction. Thus, an estimate of the earthworm NOAEL for DIMP, using the conservative acute value of 565 mg/kg as the LD50, would be 113 mg/kg (565 mg/kg ÷ 5). The hazard quotient for the earthworm would be 0.05 (6.02 mg/kg ÷ 113 mg/kg). An HQ of 0.05 indicates that DIMP is unlikely to cause an adverse ecological effect to the earthworm community. This in turn suggests that DIMP is unlikely to be a risk to other soil and litter invertebrates (Efroymson et al., 1997a).



**TABLE 5. SUMMARY OF THE DIMP HAZARD QUOTIENTS FOR THE  
POPULATION/COMMUNITY ENDPOINTS**

<b>Population/Community</b>	<b>Hazard Quotient</b>
Soil microorganisms (bacteria)	0.06
Soil and litter invertebrates	0.05
Terrestrial plants	0.60
Aquatic microorganisms (bacteria)	0.06
Aquatic algae	0.01
Aquatic invertebrates	0.04 - 0.10
Fish	0.04
Amphibians	0.02
Birds	0.63
Mammals	0.15 - 0.76

The toxicity data base for terrestrial plants is very limited for use at the Building E3640 and Kings Creek areas. It is not clear that the data for agricultural and horticultural plants can be used to assess the risk of DIMP to the plant species. The existing data are further limited because they are qualitative and most regulatory criteria are based on concentrations in toxicity tests that cause effects which are significantly different (i.e., statistically different) from controls (Efroymsen et al., 1997b).

The assumption is being made that the phytotoxicity of DIMP reported for agricultural and horticultural plants by O'Donovan and Woodward (1977a,b) will be the same for the terrestrial flora in the Building E3640 and Kings Creek areas. According to the investigators, phytotoxic symptoms in the hydroponic tests indicated that a phytotoxic effect occurred between 10 and 100 mg/L, while in soil growth experiments, no phytotoxic effects occurred at concentrations up to 20 mg/L (applied irrigation concentration) during 5-month exposures. A worst case assumption would be that the NOAEL for all terrestrial plants would be 10 mg/kg DIMP. Using 10 mg/kg as the NOAEL, the HQ for terrestrial plants would be 0.60 (6.02 mg/kg ÷ 10 mg/kg). An HQ <1 suggests that DIMP would not be a hazard to terrestrial plants. Based on the information available at this stage, the

screening-level risk data indicate that a negligible potential exists for ecological impact to microbial, invertebrate, and plant communities in direct contact with DIMP in the soil.

### 3.2.2 Adverse Effects to Aquatic Life from Exposure to DIMP in the Sediment and Water Column

As was the case for soil microorganisms, the biodegradation studies by Spanggord et al. (1979) also provide indirect evidence that DIMP is not toxic to aquatic microorganisms (bacteria) at concentrations up to 100 mg/L (Sect. 2.3.1.1). Thus, one may assume that the NOAEL would be at least 100 mg/L. The hazard quotient for aquatic bacteria exposed to DIMP would be 0.06 ( $6.02 \text{ mg/L} \div 100 \text{ mg/L}$ ). An HQ of 0.06 indicates that DIMP is unlikely to cause an adverse ecological effect to aquatic bacterial communities.

The chronic NOAEL (reduction in growth) for the green alga, *S. capricornutum*, exposed to DIMP is 711 mg/L (Table 1). The HQ for the green alga would be 0.01 ( $6.02 \text{ mg/L} \div 711 \text{ mg/L}$ ). As discussed in Section 2.3.1.4, the blue green alga, *M. aeruginosa*, is the most sensitive alga in the acute toxicity data base (Table 1). The acute to chronic ratio is frequently used as an uncertainty factor to estimate the chronic toxicity of chemicals to aquatic invertebrates and fish when only acute data are available (Kenaga, 1982). If one assumes that the ratio can also be used for aquatic algae, the acute and chronic toxicity data for *S. capricornutum* can be used to derive an uncertainty factor to estimate the chronic NOAEL for *M. aeruginosa*. The acute to chronic ratio for *S. capricornutum* is 4.5 ( $3,185 \text{ mg/L} \div 711 \text{ mg/L}$ ). Thus, the chronic NOAEL for *M. aeruginosa* would be 496 mg/L ( $2,234 \text{ mg/L} \div 4.5$ ). The HQ for *M. aeruginosa* would be 0.01 ( $6.02 \text{ mg/L} \div 496 \text{ mg/L}$ ). The HQs of 0.01 for both the green and blue-green alga indicate that DIMP is unlikely to cause an adverse ecological effect to algae.

DIMP toxicity data exist for two benthic organisms (Table 1). A recent analysis of numerical water quality criteria for nonionic organics by EPA has shown that freshwater and saltwater benthic organisms, in general, have toxicological sensitivities similar to that of water column organisms (U.S. EPA, 1993a). The assumption that benthic organisms have similar sensitivities to water column species has a level of uncertainty. For example, the tubes of some tube-dwelling amphipods tend to isolate the animals from interstitial water, causing speculation that their exposure is at the sediment/water interface (Jones et al., 1997). However, as stated in Section 3.1, the assumption is being made that the highest concentration found in the groundwater (6.02 mg/L) is in equilibrium with the bulk sediment, sediment interstitial water and water column. Thus, the toxicity data presented in Table 1 will be used to predict toxicity for both benthic and water column organisms in Kings Creek.

The lowest chronic NOAEL for invertebrates and fish exposed to DIMP is 142 mg/L for both the cladoceran (reduction in neonate production) and larval fathead minnow (reduction in growth) (Table 1). The hazard quotient for both species would be 0.04 ( $6.02$

mg/L ÷ 142 mg/L). An HQ of 0.04 indicates that DIMP is unlikely to cause an adverse ecological effect to a representative important aquatic invertebrate and fish.

The daphnid, which has a 48-h LC50 of 267 mg/L, is the most acutely sensitive species shown in Table 1. Using the uncertainty factor of 4.3, which is obtained from the acute to chronic ratios of both the cladoceran and fathead minnow, the chronic NOAEL for the daphnid would be 62 mg/L (267 mg/L ÷ 4.3). The HQ for the daphnid would be 0.10 (6.02 mg/L ÷ 62 mg/L). This indicates that DIMP is unlikely to cause an adverse ecological effect to the most sensitive species listed in Table 1 when the HQ is estimated by the acute to chronic ratio method.

Toxicity data are available for a larval frog (Table 1). The NOAEL for mortality in larval *X. laevis* is 398. The HQ for the larval frog would be 0.02 (6.02 mg/L ÷ 398 mg/L). An HQ of 0.02 indicates that DIMP is unlikely to cause an adverse ecological effect to larval frogs. As discussed in Section 2.3.1.4, the teratogenic index for the larval frog was ~1.3; thus, a low potential also exists that DIMP is a developmental hazard to frogs.

The HQ data for bacteria, algae, invertebrates, fish, and an amphibian indicate that DIMP is unlikely to be a risk to the aquatic organisms in Kings Creek. Likewise, freshwater invertebrates and amphibians that could be exposed to DIMP if the surficial aquifer moved through the surface soils into the palustrine wetland areas during wet periods of the year appear to be at low risk. The aquatic organisms in the tidal wetland areas exposed during intermittent flooding associated with normal action of the tides also do not appear to be at risk if exposed to DIMP. Based on the information available at this stage, the screening-level risk data indicate that DIMP has a negligible potential for ecological impact to aquatic communities in the Kings Creek area.

### **3.2.3 Adverse Effects to Wildlife from the Ingestion of Material Containing DIMP**

As discussed above, terrestrial wildlife may be exposed to contamination via three pathways: oral, dermal, and inhalation. Dermal exposure will be assumed to be negligible because DIMP is highly water soluble and should have a low affinity for dermal uptake relative to nonpolar organics which would have a high affinity for dermal uptake. Inhalation of DIMP is also assumed to be low because 1) the contaminant is located primarily in the subsurface soils and surficial aquifer, 2) if the compound moves to the surface soils volatilization of DIMP from soil is a slow process, and 3) the half-life of volatilized DIMP is estimated to be 5.2 h because it reacts with photochemically produced hydroxyl radicals.

Oral exposure occurs through the consumption of contaminated food (either animal or plant), drinking of contaminated water, or ingestion of contaminated soil. As discussed by Sample et al. (1997), very few wildlife consume diets that consist exclusively of one food type. To meet nutrition needs for growth, maintenance, and reproduction, most wildlife consume varying amounts of multiple food types. It is unlikely that all food types consumed will contain the same contaminant concentrations. However, for the purposes of this screening-level risk assessment, the worst case assumption will be made that the

oral exposures for all wildlife will be a maximum of 64 mg/kg (6.02 mg/kg [maximum in soil] x 10.7 [plant BCF]) for reasons discussed below.

A number of exposure-modifying factors (e.g., home range, season, behavior, etc.) can modify contaminant exposure. For the purposes of this assessment, the assumption is being made that all animals are always in contact with the contaminant at the concentration given above. Finally, the assumption is being made that individual-level exposure estimates can be used to predict population-level effects.

One subchronic oral toxicity study has been conducted with birds (Table 4). The NOAEL (body weight) for mallards exposed to DIMP for 8 d was 1,007 mg/kg/d. According to Sample et al. (1996), a chronic NOAEL for birds may be estimated from a subchronic NOAEL using an uncertainty factor of 10. A subchronic exposure duration for birds is considered to be an exposure duration  $\leq 10$  weeks, while chronic exposures are  $>10$  weeks (Sample et al., 1996). The chronic NOAEL for the mallard, when corrected by an uncertainty factor of 10, is 101 mg/kg/d. The mallard, which is primarily an aquatic herbivore/insectivore, also feeds on agricultural grains, and to a limited extent, leaves, buds, stems, rootlets, and tubers (U.S. EPA, 1993b). Thus, to be conservative in the HQ estimate, the assumption is being made that the bird feeds only on plant material which contains 64 mg/kg DIMP. The assumption is also being made that water and incidental sediment intake will contain the same concentration of DIMP. The hazard quotient for the mallard is 0.63 (64 mg/kg/d  $\div$  101 mg/kg/d). An HQ of 0.63 indicates that DIMP is unlikely to cause an adverse ecological effect to the mallard. Recent data discussed by Sample et al. (1996), suggest that physiological scaling factors developed for mammals (see below) may not be appropriate for interspecies extrapolation among birds. Sample et al. (1996) presented data which show that the scaling factor for bird body weight is  $\sim 1$ ; thus, the NOAEL data for the mallard could be used for smaller or larger birds with similar diets.

NOAELs for the mammals in the Building E3640 and Kings Creek areas will be estimated using the three generation oral rat reproduction and developmental NOAEL of 300 mg/kg/d (Table 4). As discussed by Sample et al. (1996 and 1997), studies have shown that numerous physiological functions such as metabolic rates, as well as responses to toxic chemicals, are a function of body size. Smaller animals have higher metabolic rates and usually are more resistant to toxic chemicals because of more rapid rates of detoxification. For mammals, it has been shown that if a NOAEL is available for a test species (NOAEL<sub>t</sub>), the equivalent NOAEL for a wildlife species (NOAEL<sub>w</sub>) can be estimated by using the following adjustment factor for differences in body size when the daily dose level of the test species has been normalized to the body weight of the test animal (mg/kg/d) (Sample et al., 1996):

$$\text{NOAEL}_w = (\text{NOAEL}_t)(bw_t \div bw_w)^{0.25}$$

where

$bw_t$  is the body weight of the mammalian test species, and

$bw_w$  is the body weight of the mammalian wildlife species

The mammals found in the Building E3640 and Kings Creek areas, which have a range of diets that may be contaminated with DIMP, are as follows: chipmunk (small terrestrial herbivore), grey squirrel (small terrestrial herbivore), white-tailed deer (large terrestrial herbivore), muskrat (aquatic herbivore), opossum (omnivore), and red fox (omnivore). The primary diet of the herbivores is plant material, while the diets of the omnivores are a mixture of plants and animals. Some of the animals are opportunistic feeders. For example, the muskrat's primary diet is aquatic plants; however, it will also feed on terrestrial plants as well as animals such as crayfish, fish, and amphibians (U.S. EPA, 1993b). Likewise, the composition of the plant and animal material in the diets of the red fox and opossum varies with the availability of food material. Thus, the worst case assumption is being made that all six animals eat only plant material which contains 64 mg/kg DIMP. The assumption is also being made that water and incidental soil intake will contain the same concentration of DIMP which is conservative because the DIMP concentrations in water (6.02 mg/L) and soil (4.8 or 6.02 mg/kg) would be much lower than the concentrations that are assumed to occur in plants as a result of bioconcentration (64 mg/kg).

The scaling factors and NOAELs for the six mammals, which were calculated from the above equation using the rat as the test animal, are summarized in Table 6. The hazard quotients for the six mammals are as follows: chipmunk = 0.15 (64 mg/kg/d ÷ 435 mg/kg/d); grey squirrel = 0.24 (64 mg/kg/d ÷ 270 mg/kg/d); white-tailed deer = 0.76 (64 mg/kg/d ÷ 84 mg/kg/d); muskrat = 0.30 (64 mg/kg/d ÷ 213 mg/kg/d); opossum = 0.42 (64 mg/kg/d ÷ 153 mg/kg/d); and red fox = 0.40 (64 mg/kg/d ÷ 159 mg/kg/d). The HQs range from 0.15 to 0.76 which indicate that DIMP is unlikely to cause an adverse ecological effect to the mammals that forage in the Building E3640 and Kings Creek areas.

**TABLE 6. BODY SIZE SCALING FACTOR AND NOAEL CALCULATED  
FROM THE RAT FOR MAMMALS<sup>a</sup>**

Species	Body Weight (bw <sub>w</sub> in kg)	Scaling Factor (bw <sub>t</sub> ÷ bw <sub>w</sub> ) <sup>0.25</sup>	NOAEL <sub>w</sub> <sup>b</sup> (NOAEL <sub>t</sub> )(Scaling Factor)
Chipmunk	0.08 <sup>c</sup>	1.45	435
Grey squirrel	0.53 <sup>c</sup>	0.90	270
Muskrat	1.36 <sup>c</sup>	0.71	213
Opossum	5.0 <sup>c</sup>	0.51	153
Red fox	4.5 <sup>d</sup>	0.53	159
White-tailed deer	56.5 <sup>e</sup>	0.28	84

<sup>a</sup> Rat body weight (bw<sub>t</sub>) = 0.35 kg (Sample et al., 1996); NOAEL<sub>t</sub> = 300 mg/kg/d (Hart, 1980).

<sup>b</sup> Each NOAEL<sub>w</sub> is expressed as mg/kg/d.

<sup>c</sup> Body weight taken from Burt and Grossenheider (1976).

<sup>d</sup> Body weight taken from Storm et al. (1976).

<sup>e</sup> Body weight taken from Smith (1991).

## 4. CONCLUSIONS

Two exposure pathways exist by which DIMP may reach the ecological receptors in the Building E3640 and Kings Creek areas. The first is the contaminated subsurface soil located in the spill area in the northeast corner of the Building E3640 site. The second potential exposure pathway is the apparent plume of DIMP which is migrating in the surficial aquifer from the Building E3640 area north northeast towards Kings Creek. All activities at Building E3640 which contaminated the soils and surficial aquifer were stopped in 1978. No DIMP is currently stored on site. Thus, no further releases of DIMP at the site will occur. Additional releases of DIMP to the groundwater could occur via the subsurface soils (infiltrating precipitation) in the spill area.

The following endpoints were used to evaluate the ecological risk of DIMP to the populations/communities in the Building E3640 and Kings Creek areas: 1) adverse effects to microorganisms, invertebrates, and plant communities from direct contact with DIMP in the soil; 2) adverse effects to aquatic life from exposure to DIMP in the sediment and water column; and 3) adverse effects to wildlife from the ingestion of material containing DIMP.

All NOAEL data used in the screening-level ecological risk assessment were based on conservative or worst case assumptions. A major assumption was made that the surficial aquifer would ultimately transport DIMP to the palustrine wetlands and tidal wetlands of the Kings Creek area as well as Kings Creek. No evidence exists which shows that DIMP has in fact moved from the Building E3640 site. Likewise, the assumption was made that DIMP concentrations in the surface and subsurface soils in the palustrine wetland areas and tidal wetland sediments were the same as the highest concentration in the surficial aquifer since the surficial aquifer would be the source of the contaminant. To be conservative, 6.02 mg/kg was used as a worst case for all soil calculations even though the highest DIMP concentration in the subsurface soils at the Building E3640 site was 4.8 mg/kg. The bioconcentration data for plants (agricultural plants) reported in the literature and used in the risk assessment are questionable (data not treated statistically). However, the highest BCF (10.7) reported in the literature for agricultural plants was used for all plants in the risk assessment. The worst case assumption was made that DIMP was bioconcentrated to 64 mg/kg dry weight ( $10.7 \text{ [BCF]} \times 6.02 \text{ mg/kg} = 64$ ) in the roots, stems, and leaves of all plants in the Building E3640 and King Creek areas.

The assumption was made that the highest concentration of DIMP found in the surficial aquifer (6.02 mg/L) at the Building E3640 site was in equilibrium with the bulk sediment, sediment interstitial water and water column in Kings Creek. Thus, any organism present in Kings Creek was assumed to be exposed to a maximum concentration of 6.02 mg/L. The worst case assumption was made that the oral exposures for all wildlife in both the Building E3640 and Kings Creek areas would be at a maximum of 64 mg/kg/d. All wildlife species found in the study area consume some plant material in their diet. Species which consume multiple food types, such as, a mixed diet of plants and animals



would not consume DIMP at a rate of 64 mg/kg/d because animals do not accumulate DIMP above background levels.

A number of exposure-modifying factors (e.g., home range, season, behavior, etc.) can modify wildlife contaminant exposure. The assumption was made that all organisms were always in contact with the contaminant at the maximum concentrations given above. The assumption was also made that DIMP was 100% bioavailable to all receptors at all times. DIMP is not expected to accumulate above background in animals because of its low  $\log K_{ow}$  (1.03); thus, DIMP bioconcentration/bioaccumulation in animals was not considered in the risk characterization. When chronic NOAEL data were not available for use in the HQ calculations, established uncertainty factors were used to estimate NOAELs from subchronic values.

No data are available regarding the modes of DIMP toxicity to soil and aquatic bacteria, soil invertebrates, terrestrial plants, aquatic algae, aquatic invertebrates, fish and amphibians; few data are available for birds. Toxicokinetic studies have shown that DIMP is rapidly absorbed (15 min to 3 h depending on the species) following oral administration in mammals. DIMP is initially distributed throughout the body via the circulatory system, followed by high concentrations primarily in the liver, kidneys, and urinary bladder in 4 to 24 h after oral administration. DIMP is metabolized primarily to IMPA; some hydrolysis of IMPA to MPA may occur in the liver or in other tissues. Peak urinary excretion of a single dose occurs between 6 and 72 h depending on the species. No storage of DIMP, IMPA, or MPA occurs in the tissues of mammals, although portions of [ $^3\text{H}$ ]-label may become incorporated in biomolecules leading to some retention of label in the form of unextractable labeled compound. DIMP is not genotoxic or carcinogenic to birds or mammals (including humans) after oral exposure. DIMP is not a developmental hazard to larval frogs.

The screening-level risk calculations show that DIMP poses a negligible risk to plants and animals in the Process Laboratory and Kings Creek areas. The conclusion is based on the fact that the HQs of all potential receptors in the study area are  $<1$ . The HQs for soil microorganisms, soil and litter invertebrates, and terrestrial plants were estimated to be 0.06, 0.05, and 0.60, respectively. The HQs for aquatic microorganisms (bacteria), aquatic algae, aquatic invertebrates, fish, and amphibians were 0.06, 0.01, 0.10, 0.04, and 0.02, respectively. The HQs for birds and mammals were 0.63 and 0.76, respectively. No DIMP data were available for reptiles; thus, an HQ was not calculated for reptiles. Based on the data for other vertebrates, the weight-of-evidence suggests that DIMP will not be a risk to reptiles.



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